NASA TECHNICAL MEMORANDUM

NASA TM X-1285

EFFECTS OF ACUTE AND CHRONIC HYPOHYDRATION ON TOLERANCE TO $+G_Z$ ACCELERATION IN MAN:

I. PHYSIOLOGICAL RESULTS

by John E. Greenleaf, M. Matter, Jr., L. G. Douglas, S. A. Raymond, J. S. Bosco, E. G. Averkin, and R. H. St. John, Jr.

Ames Research Center

Moffett Field, Calif.

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION • WASHINGTON, D. C. • SEPTEMBER 1966

NASA TM X-1285

EFFECTS OF ACUTE AND CHRONIC HYPOHYDRATION ON TOLERANCE TO $+G_Z$ ACCELERATION IN MAN: I. PHYSIOLOGICAL RESULTS

By John E. Greenleaf, M. Matter, Jr., L. G. Douglas, S. A. Raymond, J. S. Bosco, E. G. Averkin, and R. H. St. John, Jr.

Ames Research Center
Moffett Field, Calif.

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION

For sale by the Clearinghouse for Federal Scientific and Technical Information
Springfield, Virginia 22151 – Price $2.00

TABLE OF CONTENTS

EFFECTS OF ACUTE AND CHRONIC HYPOHYDRATION ON TOLERANCE TO $+G_Z$ ACCELERATION IN MAN: I. PHYSIOLOGICAL RESULTS

By John E. Greenleaf, M. Matter, Jr., L. G. Douglas, S. A. Raymond, J. S. Bosco, E. G. Averkin, and R. H. St. John, Jr.
Ames Research Center

SUMMARY

Two groups of male subjects were hypohydrated approximately 3.6 percent of their total body weight either by means of a sauna bath (acute group) or a 48-hour water restriction period (chronic group). Following hypohydration each group underwent four centrifugation runs at an acceleration build-up of +3.7 G/min - held at 6.0 G until blackout occurred. The results indicated (a) no significant difference in mean tolerance times between the acute and chronic group; (b) a significant decrease ($p < 0.005$) in mean tolerance times between the normohydration and hypohydration groups; and (c) a significant decrease ($p < 0.001$) in mean tolerance times over the four successive runs. The mechanisms of reduced tolerance to $+G_Z$ acceleration when hypohydrated are complex because there was very little relationship between percent body weight loss, red cell volume, plasma volume, and total blood volume and tolerance time. The concept of free circulating water was advanced as a possible explanation for the conflicting results regarding the effects of water depletion on tolerance to $+G_Z$ acceleration.

INTRODUCTION

Man's tolerance to acceleration remains crucially important in aerospace medicine where physiological tolerance limits have increasing application as engineering constraints during the development of high performance aircraft and space vehicles.

Since the preliminary observations of Diringshofen (ref. 1), Livingston (ref. 2), and Andina (ref. 3), a burgeoning literature has attempted to delineate the physiological mechanisms associated with $+G_Z$ acceleration tolerance.[1]

[1]In some cases in the literature there appears to be inadequate definitions of experimental conditions in regard to the subject's position in the centrifuge. This paper employs a convention discussed by Webb (ref. 4) and modified to emphasize the expression of acceleration in gravitational units rather than conventional velocities/sec or dx^2/dt^2 notations. A capital G rather than a lower case g is used to denote the gravitational unit of acceleration to avoid confusion with the accepted symbol g for gram (see ref. 5 for such a mistake).

Primarily, the impetus for these studies has centered around practical considerations. The general aim has been to investigate the effects of mechanical (refs. 6-10) and physiological (refs. 11-15) variables on acceleration tolerance.

In February 1962 a comparatively new and important variable in acceleration tolerance emerged. Despite ad libitum availability of drinking water during the four orbital flights of Project Mercury (refs. 16-19), each of the four astronauts returned with symptoms indicative of hypohydration. The term "hypohydration" is used here as a more general expression for a water deficit than the term "dehydration," which connotes a total absence of water. Also, the term "normohydration" refers to the normal, ad libitum water consumption. The terminology follows the suggestions of Johnson (ref. 20).

This study was carried out during July 1964 in direct response to the practical concern for the effects of hypohydration on tolerance to acceleration.

However, it was also likely that the question of hypohydration and effects on $+G_z$ tolerance would prove to be of basic physiological interest. Results of the numerous studies consistently indicate that the "Achilles heel" of human $+G_z$ acceleration tolerance is the cardiovascular system (refs. 3, 21-25). Alterations in water balance, body water content, blood volume, and blood viscosity as a result of hypohydration usually change the functioning of the cardiovascular system (refs. 11, 26-30). This is particularly evident during the strain of acceleration. In this study we investigated the effects of hypohydration induced by water deprivation or heat stress on tolerance to $+G_z$ acceleration.

PROCEDURE AND METHODS

General Experimental Plan

This study was divided into three experiments (table 1): N1, normohydration; H2, hypohydration; and N3, normohydration. During N1 and N3 the subjects

TABLE 1.- EXPERIMENTAL SCHEDULE, JULY 1964

Acute hypohydration group				Chronic hypohydration group			
Subject	N1a	H2a	N3a	Subject	N1c	H2c	N3c
MM	1 July	23 July	30 July	DD	8 July	24 July	31 July
HV	2 July	20 July	30 July	JB	8 July	21 July	28 July
WL	6 July	20 July	27 July	JL	16 July	22 July	29 July
RP	6 July	20 July	29 July	MP	17 July	21 July	28 July
JM	23 July	24 July	31 July	DL	17 July	22 July	29 July
				CO	17 July	22 July	29 July
				FK	21 July	24 July	31 July
				JG	2 July	16 July	23 July

consumed water ad libitum and ate their normal diet. In the H2 experiment the subjects were divided into two groups: acute hypohydration group (H2a), and chronic hypohydration group (H2c); controlled diets and programmed water intakes were prescribed for the subjects. Each subject rode the centrifuge three times: N1, H2, and N3.

Subjects

The subjects were 13 healthy men, aged 22 to 36 (table 2). Two of them (subjects MM and WL) were experienced on the centrifuge; the others were essentially novices. The two experienced subjects were in the acute group.

TABLE 2.- ANTHROPOMETRIC AND PHYSIOLOGIC BASELINE DATA ON THE SUBJECTS

Subject	Occupation	Age	Height, cm	Weight, kg	S.A., m^2	Normo-hydration blood vol., ml	Lying pulse rate, beats/min	Hypo-hydration regime
MM	Pilot-Physician	32	181.0	76.395	1.97	6326	60	Acute
HV	Engineer	27	182.2	68.700	1.88	6824	81	Acute
WL	Pilot	33	177.2	73.610	1.90	5870	54	Acute
RP	Physician	32	164.5	69.955	1.76	6432	50	Acute
JM	Student	25	171.1	65.055	1.76	6313	60	Acute
DD	Student	25	179.7	72.425	1.90	8052	44	Chronic
JB	Professor	36	165.7	66.340	1.74	6290	58	Chronic
JL	Student	22	161.9	60.050	1.64	5422	62	Chronic
MP	Student	22	170.2	66.485	1.77	5995	64	Chronic
DL	Student	22	175.9	70.490	1.85	6396	74	Chronic
CO	Student	22	177.8	73.975	1.90	6601	52	Chronic
FK	Teacher	29	176.5	77.065	1.94	6342	60	Chronic
JG	Scientist	31	177.2	78.085	1.96	5782	66	Chronic

Hypohydration Regimen and Diet

A sauna bath (50° to 80° C) was utilized to hypohydrate the subjects in the acute hypohydration experiments. The subjects stayed in the sauna bath about 3 to 4 hours until they had lost about 2.5 kg of body weight (table 3).

TABLE 3.- BODY WEIGHT CHANGES DURING THE EXPERIMENTAL PERIODS

	N1 kg	H2 Pre wt, kg	H2 End wt, kg	H2 N1 - H2 end, kg	H2 Wt loss, percent	H2 Wt loss, g/hr	H2 Wt loss, g/hr - m^2	N3 kg
Acute group								
$\bar{X}$	70.743	70.069	68.351	2.392	3.36	502	272	70.082
±SE	1.97	1.73	1.79	0.254	0.301	52.68	31.04	1.78
Chronic group								
$\bar{X}$	70.614	71.486	67.879	2.735	3.79	76	41	70.338
±SE	2.15	2.02	1.82	0.474	0.577	6.72	3.11	1.98

At least one hour elapsed between their leaving the sauna bath and entering the centrifuge. No food or water was allowed after the pre-sauna body weight was measured until the experiment (centrifugation) was completed.

Chronic hypohydration (table 3) was achieved by placing the subjects on a controlled diet (table 4) for approximately 48 hours. The diet consisted of

TABLE 4.- FOOD CONSUMPTION BY THE CHRONICALLY HYPOHYDRATED SUBJECTS (per 48 hours)

Subject	Metrecal[a] wafers, kcal	Metrecal[b] liquid, kcal	Water,[c] ml	$C_{12}H_{22}O_{11}$, g	Sugar[d] ingestion, min	Sugar[e] ingestion, min	Sodium intake, g
DD	1800	900	1048	38	1	55	2.9
JB	1800	900	1048	38	19	50	2.9
JL	900	900	1048	38	-	-	1.9
MP	850	900	1048	38	18	51	1.8
DL	1800	900	1048	38	4	38	2.9
CO	1800	900	1048	38	24	57	2.9
FK	425	900	1048	38	32	130	1.4
JG	1425	900	1048	38	37	98	2.5
$\bar{X}$	1350	900	1048	38	19	68	2.4

[a]Metrecal wafers - 25 kcal/wafer
[b]Metrecal liquid - 225 kcal and 237 ml H_2O/8 oz can
[c]4 cans Metrecal liquid = 948 + 100 ml with sugar
[d]Time between sugar ingestion and blood sample
[e]Time between sugar ingestion and centrifugation

Metrecal liquid and Metrecal wafers - a total of 1350 kcal and 474 ml of fluid per day (see tables VII and VIII). In addition to restricting water, this was also a semistarvation diet. Thus, part of the weight loss in the H2c group was probably due to a caloric deficit. Prior to centrifugation (about 68 min) each H2c subject drank a mixture consisting of 38 g of table sugar in 100 ml of tap water (table 4) to assure that there was no hypoglycemia (ref. 15). In addition, subjects JM and MM (acute group) took 38 g of sugar 20 minutes prior to centrifugation. No other food or water was allowed until the experiment was completed. The H2c subjects carried on their normal daily activities during the 2-day hypohydration period.

Each experiment (table 5) lasted approximately 2 hours and as many as 4 subjects were tested on any 1 day (table 1). The H2a subjects that were hypohydrated in the sauna bath in the morning rode the centrifuge that same afternoon. The H2c subjects began their diet in the afternoon and rode the centrifuge approximately 48 hours later. Thus, both hypohydration groups rode in the afternoon.

TABLE 5.- TYPICAL EXPERIMENTAL SCHEDULE

Time	
0908	Urine sample
0912	Body weight
0914	Resting
0930	Pulse rate - lying
0931	Blood pressure - lying
0932	Blood pressure - standing
0934	Blood pressure - standing (2 min)
0934	Pulse rate - standing (2 min)
0936	Pre-dye control blood sample
0936	Inject Evans blue dye
0946	Post-dye blood sample
0950	Hookup for centrifugation
1038	Ride centrifuge
1050	Egress from centrifuge cab
1115	Questioning regarding subjective feelings, etc.

Centrifuge

The centrifuge was the Ames five-degree-of-freedom motion simulator (ref. 31). The single-place cab, supported inside a gimbaled structure, was mounted on an arm with a 9.1 m (30 ft) radius (fig. 1). The subjects were restrained in a standard aircraft seat with a lap and chest harness. The helmet was secured so the subject's head would not be displaced. The roll of the cab was controlled by electronic computers so the acceleration vector was maintained parallel to the long axis of the body.

The pulse rate (electrocardiogram) and oxygen saturation (ear oximeter) were included in the measurements taken during centrifugation (fig. 2). An intercommunication system was used between the subject and medical monitor and a closed circuit television monitor was used to observe the subject's face.

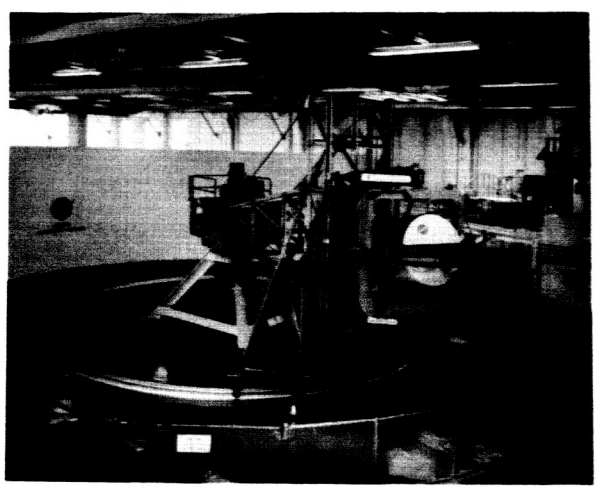

Figure 1.- Ames five-degrees-of-motion simulator.

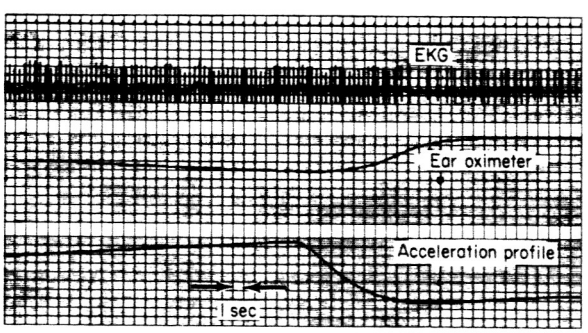

Figure 2.- Typical centrifugation record.

On the test day, each subject underwent four successive runs at an acceleration buildup of 3.7 G/min up to 6.0 G, the machine's maximum, and this level was held until blackout occurred. There was a 1 to 1-1/2 minute rest period between each run. Sphygmomanometric blood pressures were measured semiautomatically during the rest periods. We followed the nomenclature of Howard (ref. 32), who delineated three stages of visual response to $+G_z$ acceleration: (1) grayout, a veiling or misting of vision; (2) blackout, complete loss of vision; and (3) unconsciousness. One central light with the luminance adjusted for blackout at about 6.0 G was positioned directly in front of the subject's eyes (ref. 33). Peripheral lights were also employed, but central blackout was used for assessing tolerance due to the apparent inaccuracies that have accompanied peripheral grayout as an end point (ref. 34).

Prior to the start of the experiments each subject had made at least 4 runs on the centrifuge for practice and orientation. The two most experienced subjects were in the acute group and the results should be interpreted accordingly. They were requested to compensate by muscular contraction for the pooling of blood in the lower extremities. All the subjects stated they compensated to some degree; none rode passively (see ref. 35).

The subjects had about 5 minutes to become accustomed to the darkness in the cab before starting an experiment. The subjects were instructed to release a deadman switch when the central light faded out; each run was terminated by the subject. At the end of each run the cab stopped with the subject on his right side. The cab was then slowly rolled upright to prevent vertigo, nausea, etc. None of the subjects became ill during or after centrifugation in the H2 and N3 experiments, but several reported feelings of gastric distress. Immediately upon egress from the cab, each subject was questioned regarding his feelings, etc., concerning the centrifugation. Those results were reported in the companion paper (ref. 35).

Biochemical Analyses

Venous antecubital blood specimens and urine specimens were collected prior to centrifugation (table 5). Evans blue space (plasma volume) was measured utilizing one 10-minute post-dye blood sample (ref. 36). Sodium heparinate was used in the pre-dye control blood sample. A correction factor of 0.96 was used to estimate trapped plasma in the capillary microhematocrit determinations (ref. 37). The following additional measurements were made on serum from the pre-dye control sample: serum glucose using glucose oxidase (ref. 38); urinary and serum total osmolarity (Fiske osmometer model Mark III); urinary and serum Cl (ref. 39); and urinary and serum Na and K (Baird flame photometer model KY-2).

Statistical Analysis

Analysis of variance was used to test for significant differences between methods of hypohydration (acute and chronic) and states (normohydration and hypohydration). A split-plot design was used, where the main plots were the

two methods of hypohydration and subplots were tolerance times. The H2 data were compared to the N3 data. Since there was some discrepancy in the acceleration profiles applied during N1, these data were not used in the statistical analysis, except for body weight. However, the N1 data are included in tables I through IV for reference. An explanation of the analysis of variance nomenclature is presented in table 6. Throughout the paper all regression lines were fitted by the method of least squares.

TABLE 6.- CENTRIFUGE TOLERANCE TIMES; ANALYSIS OF VARIANCE

Source	Degree of freedom	Sum of squares	Mean square	F	p
Between subjects					
Method	1	9.663	9.663	0.010	n.s.
Error (a)	11	11,684.816	1,062.256		
Within subjects					
State	1	5,016.512	5,016.512	12.404	< 0.005
M × S	1	44.758	44.758	0.111	n.s.
Error (b)	11	4,448.641	404.422		
Time	3	2,218.091	739.364	6.809	< 0.001
T × M	3	275.626	91.875	0.846	n.s.
Error (c)	33	3,583.117	108.579		
S × T	3	254.410	84.803	0.851	n.s.
M × S × T	3	148.007	49.336	0.495	n.s.
Error (bc)	33	3,287.380	99.618		

Method	acute versus chronic
State	normohydration versus hypohydration
Time	comparison of the four centrifuge runs
Error (a)	subjects within method
Error (b)	subjects within method × state
Error (c)	subjects within method × time
Error (bc)	subjects within method × state × time
n.s.	not significant at $p < 0.05$

RESULTS

Centrifuge Tolerance

The results of the analysis of variance (table 6) indicated: (1) no significant difference in mean tolerance times between the acute and the chronic groups; (2) a significant decrease ($p < 0.005$) in mean tolerance times between the normohydration and hypohydration groups; and (3) a significant decrease ($p < 0.001$) in mean tolerance times during the four successive runs (the fatigue effect). These results are shown graphically in figure 3. In

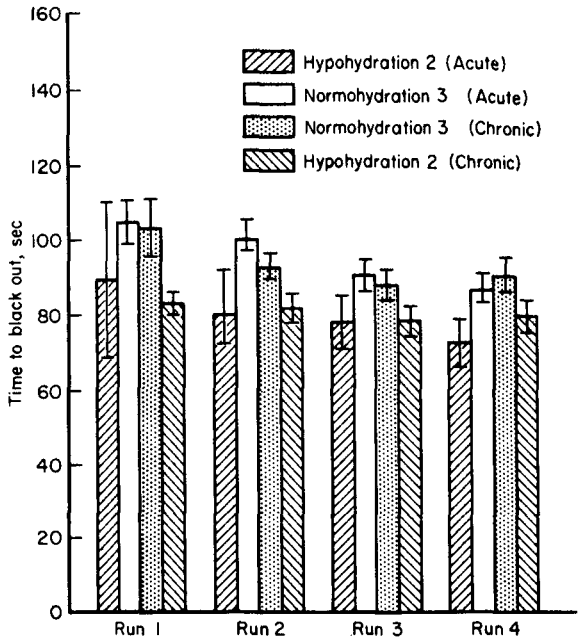

Figure 3.- Average tolerance times for the various experimental groups.

interpreting these results, one must keep in mind that the acute group might have had some residual effects of the heat per se and that the chronic group were in caloric deficit and were hyperglycemic when they rode the centrifuge. The differences in tolerance times indicated that most of the fatigue decrement occurred during or after 2 runs with a lesser decrement between the third and fourth runs. The H2c group (table 7) had the lowest average tolerance time (run 1), and there was very little decrement (3.5 percent) between runs 1 and 4. The decrements in the acute hypohydration groups (H2a - 18.2 percent vs N3a - 17.2 percent) were about equal. In run 1, the H2a group had a lower tolerance time than the N3a group. Thus, acute hypohydration reduced tolerance time but did not appreciably change the fatigue effect. The chronic group also exhibited a decrease in tolerance time when group H2c (82.9 sec) was compared with N3c (103.8 sec) in run 1, but group H2c (the hyperglycemic group) had a much smaller fatigue effect (3.5 percent) compared with N3c (12.3 percent).

TABLE 7.- TOLERANCE TIMES AND THE FATIGUE EFFECT DURING CENTRIFUGATION (seconds)

	H2a	N3a	Percent decrement	H2c	N3c	Percent decrement
Run 1	89.5	105.1	14.8	82.9	103.8	20.1
Run 4	73.2	87.0	15.9	80.3	91.0	11.8
Percent decrement	18.2	17.2		3.5	12.3	

Heart Rate Changes During Centrifugation

Heart rates were counted during the 10-second period immediately before centrifugation (pre) and during the 10-second period during deceleration (post). The average changes in heart rates (post minus pre) for the various experimental groups are presented in figure 4. The post-centrifugation average heart rates are also plotted directly above their respective groups. With the exception of the H2a group, whose average differences in heart rates were essentially equal for the first three runs, there was a progressive diminution in the differences during the first two runs and then a leveling off during runs 3 and 4. The general tendency of the differences in the heart rates to

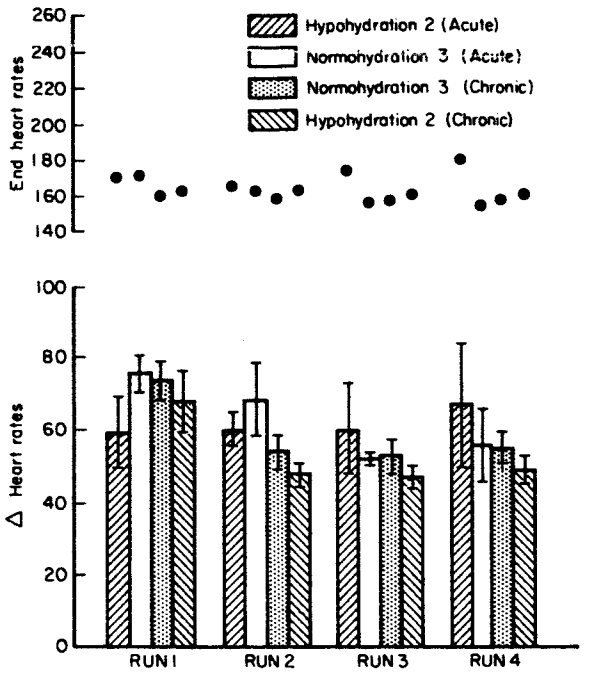

Figure 4.- Heart-rate changes for the various experimental groups during centrifugation.

decline during successive runs paralleled a similar decline in tolerance times (fig. 3). In general, the differences in the heart rates (run 1 through run 4) were due to a slight progressive increase in the pre-run heart rate and a slight decrease in the post-run rates. However, the H2a group showed the opposite effect - increased post-run rates and relatively constant pre-run heart rates. It appeared that the centrifugation produced more strain in the H2a group since the post-run pulse rates were higher than in the other groups. The H2c group followed the pattern of the N3c and N3a groups. It was coincidental, but interesting, that there was a 6-beat/min difference between H2c and N3c during all 4 runs.

Body Weight Changes and Tolerance Times

A comparison of the H2 and N1 body weights after hypohydration indicated that the acute group (H2a) was 3.4-percent hypohydrated and the chronic group (H2c), 3.8 percent (fig. 5). The N1 body weight was selected as the basis for the calculation of hypohydration rather than N3 because N1 was the more accurate "normal" body weight. The N3 mean body weights for the two groups were slightly lower than the corresponding N1 weights, perhaps indicating some residual caloric deficit and/or voluntary dehydration (ref. 28). Since the mean body weights for the two groups were approximately the same in N1, H2 post-hypohydration, and N3, it appeared that the wide difference between the body weights of the two groups observed in H2 pre-hypohydration (fig. 5) may have resulted from increased amounts of food and/or water consumption in expectation of the forthcoming dietary restriction period. Therefore, it seemed appropriate to compare H2 post-hypohydration with N1 when the percent body weight loss was computed. Table VI presents additional data regarding body weight changes.

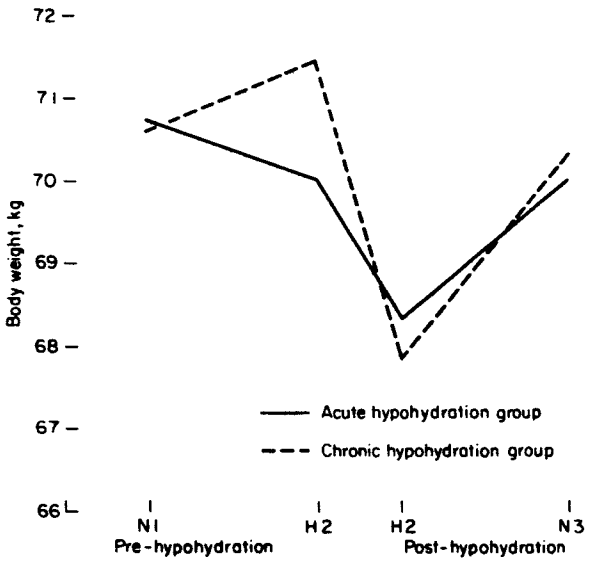

Figure 5.- Body weight changes with hypohydration.

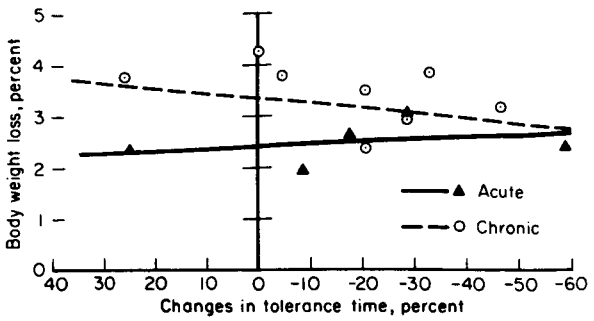

Figure 6.- Body weight loss and tolerance times.

There was essentially no relationship between body weight loss and run 1 tolerance time decrements in H2 (fig. 6). However, body weight loss is not an accurate measurement of hypohydration. Also, the diet caused a caloric deficit in the chronic group.

Cardiovascular and Metabolic Variables

The significant changes in the cardiovascular and metabolic variables between method (acute vs chronic) and state (normohydration vs hypohydration) are presented in table 8. (See table V(b) for the complete results of the

TABLE 8.- CARDIOVASCULAR AND METABOLIC VARIABLES; ANALYSIS OF VARIANCE SUMMARY

	Method	Error (a)	State	Method × State
Lying pulse rate	-	-	-	-
Standing pulse rate	-	< 0.050	< 0.001	-
S - L pulse rate	-	< 0.025	< 0.001	-
Lying SBP	-	< 0.001	-	-
Standing SBP	-	< 0.025	< 0.050	-
S - L SBP	-	-	< 0.050	-
Lying DBP	-	< 0.005	-	-
Standing DBP	-	< 0.005	-	-
S - L DBP	-	-	-	-
2-min standing SBP	-	< 0.025	-	-
Δ standing SBP	-	-	-	-
2-min standing DBP	-	-	-	-
Δ standing DBP	-	-	-	-
Serum glucose (mg/100 ml)	< 0.05	-	< 0.005	-
Evans blue space (ml)	-	< 0.01	< 0.001	-
Red cell volume (ml)	-	< 0.001	< 0.025	< 0.01
Blood volume (ml)	-	< 0.001	< 0.001	-
Hematocrit (vol percent)	-	< 0.001	< 0.010	-
Serum Na (mEq/l)	-	-	-	-
Serum K (mEq/l)	< 0.025	-	< 0.05	-
Serum Cl (mEq/l)	-	-	-	-
Serum osmolarity (mOsm/l)	-	-	< 0.01	-
Urine volume (ml)	-	< 0.01	-	< 0.05
Urine Na (mEq/l)	-	< 0.025	< 0.001	< 0.005
Urine K (mEq/l)	-	-	< 0.005	-
Urine Cl (mEq/l)	-	< 0.05	< 0.001	< 0.01
Urine osmolarity (mOsm/l)	< 0.05	-	< 0.025	-

analysis of variance.) Only serum glucose, serum K, and urine osmolarity were significantly different in the acute and chronic hypohydration groups. Since sugar was given to the chronic group, the difference in serum glucose was to be expected.

On the other hand, many variables in the normohydration group differed significantly from those in the hypohydration group. Some orthostatic intolerance was present under hypohydration. The immediate standing pulse rate increased 20 beats/min, and the immediate standing minus lying pulse rate increased 15 beats/min. The immediate standing systolic blood pressure (SBP) decreased 10 mm Hg as did standing minus lying systolic blood pressure. Neither the lying nor the 2-minute standing pulse rate, nor systolic nor diastolic blood pressure, changed with hypohydration. The 10 mm Hg decrease in standing SBP was essentially a normal response, but the 20-beat/min increase was about twice the normal increment when the subject assumed the upright position (ref. 40). Serum glucose was different for the reason mentioned above. All the constituents of the blood volume changed significantly with hypohydration: the plasma volume decreased 410.1 ml, the red cell volume decreased 116.4 ml, the total blood volume decreased 526.5 ml, and the hematocrit increased 1.8 volume percent. The reason for the discrepancy between the red cell volume and hematocrit is not readily apparent. The serum Na and Cl were unchanged, while the serum K and osmolarity were significantly increased with hypohydration. The urinary Na and Cl were significantly decreased and the urinary K and osmolarity were increased, the latter two variables reflecting the elevated serum levels.

In table 8 error (a) indicates the level of significance of the intraclass correlation coefficient $\hat{\rho}$. A significant error (a) means that the value of a particular variable in H2 was highly correlated with the N3 value for the same individual. A significant value in the last column indicates interaction between method and state. This means that the relationship between the values of a particular variable (i.e., serum sodium) for groups N3c and H2c was either not in the same direction or not of the same magnitude as the same variable in groups N3a and H2a, indicating a differential response to hypohydration and the method used to induce it. From table 8 it can be seen, under Method × State, that red cell volume, urinary volume, urinary sodium, and urinary chloride exhibited interaction (see fig. 7). Conversely, the urinary sodium and chloride values paralleled each other very closely in the chronic and in the acute experiments. A very high correlation (r = 0.97) was found in previous work between the mean daily urinary sodium and chloride (ref. 41).

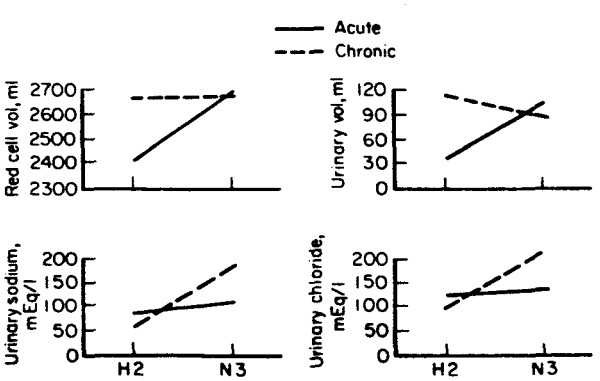

Figure 7.- Significant method × state interactions - analysis of variance.

Blood Volume Constituents and Tolerance Times

The red cell volume, Evans blue space (plasma volume), and total blood volume for the four experiments are presented in figure 8. The red cell volumes were essentially equal in N3a, H2c, and N3c, and about 250 ml lower in H2a. In general, the hypohydration plasma volumes were about 400 ml less than the normohydration plasma volumes. The H2a total blood volume was about 750 ml less than N3a; H2c was about 400 ml less than N3c. Thus, the total blood volume for the acute group was decreased about twice as much as the chronic group. Since the levels of total body weight loss reached by the two hypohydration groups were essentially equal, it is likely that the greater total blood volume loss of the acute group was due to the nature of the hypohydration process - short time plus intense heat - as has been described by Saltin (ref. 29).

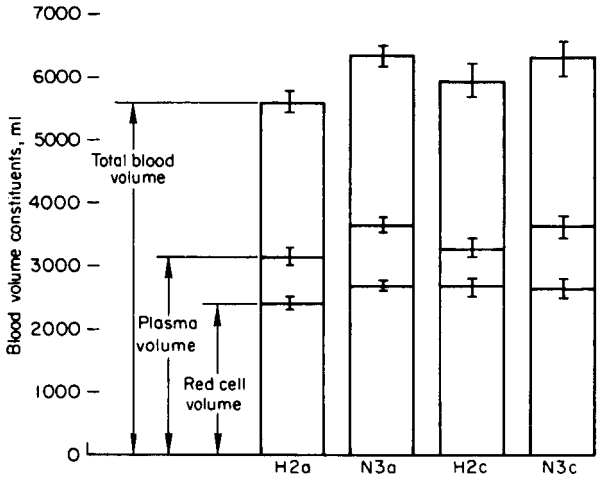

Figure 8.- Blood volume constituents in normo- and hypohydration.

There was little, if any, relationship between the percent blood volume loss and percent change in run 1 tolerance time in either the acute or chronic groups (fig. 9). The plasma volume also exhibited very little relationship to run 1 tolerance time in the two hypohydration groups (fig. 10 - left side). The two normohydration groups (fig. 10 - right side) showed a negative relationship between plasma volume and run 1 tolerance time. Since the sample sizes are rather small, no definite conclusions can be drawn. From the evidence it appears that there is no definite positive relationship between the decrease in the blood volume constituents and decrements in tolerance time.

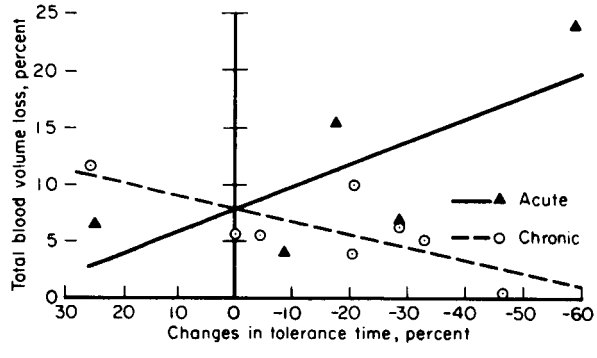

Figure 9.- Blood volume changes and tolerance times.

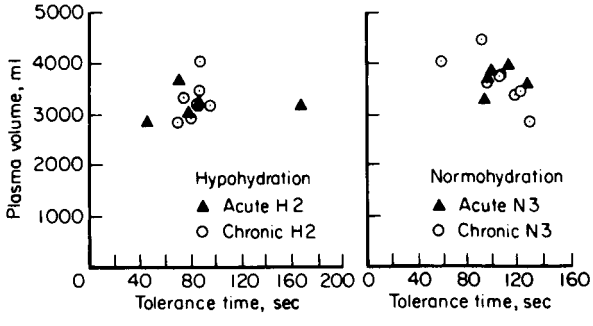

Figure 10.- Plasma volumes and tolerance times.

DISCUSSION

Before the mechanisms involved in the decreased tolerance of hypohydrated subjects can be discussed, the problem must be clearly defined. Blackout

tolerance, as used in this study, is essentially the point when the subject can no longer see the central light. Presumably, at that point the blood pressure in the retinal vessels drops to about 20 mm Hg where the excess intraocular pressure compresses the arteries completely, and vision is lost even though the blood pressure in the rest of the brain is adequate (refs. 3, 32, and 42). Thus, the "final common pathway" in the chain of events influencing blackout tolerance is the maintenance of the retinal arterial blood pressure, the latter being a reflection of the systemic blood pressure as a whole. In this study we were looking at the effects of an increasing hydrostatic pressure head due to centrifugation plus the effects of muscular exercise (essentially intermittent isometric contractions) plus the influence of two types of hypohydration on the ability of the physiological systems to maintain blood pressure, specifically in the retinal vessels.

The complex dynamic regulation of the blood pressure during acceleration depends upon two physiological variables; cardiac output (flow) and peripheral resistance according to the general formula:

(1) Pressure = Flow × Resistance, or

(2) Blood pressure = Cardiac output × Peripheral resistance

If hypohydration alters blackout tolerance through a physiological mechanism or mechanisms, it probably acts by modifying cardiac output and/or peripheral resistance. Three main systems serve to control mean arterial pressure. Categorized on a time basis they are: (1) the nervous system reflexes which act 5 to 10 seconds; (2) the capillary fluid shift that takes 2 to 30 minutes; and (3) the kidney pressor substances that are the slowest acting and take from 2 to 3 days to act. Since each run lasted no longer than 2-1/2 minutes, the kidney mechanism probably was not important. We will discuss the nervous reflexes and the capillary fluid shift mechanisms in more detail.

The nervous system reflexes operate through the sympathetic nervous system which functions to alter blood vessel diameter and influences the heart rate and force of contraction. One very important reflex during acceleration is the carotid sinus reflex which responds to changes in arterial pressure. A drop in pressure, such as occurs during $+G_z$, causes a decrease of stretch tension in the walls of the carotid receptor. This lowered tension leads to an increase in autonomic vasoconstrictive activity and a decrease in vasodilatory activity; the final effect is vasoconstriction and increased heart rate and strength of contraction (ref. 43). Other known neurotensive systems related to the control of blood pressure, such as the increase in CO_2, the lack of O_2, and the changes in blood volume that follow, are probably less important during $+G_z$ than the carotid sinus reflex because centrifugation is primarily a mechanical rather than a biochemical stress. However, the hormones of the adrenal medulla, which can reflect emotional levels, exert profound effects on heart rate and blood pressure and must not be overlooked when mechanisms influencing tolerance to $+G_z$ acceleration are considered. It is plausible that the nervous reflexes play the major role in maintaining blood pressure during the acceleration.

Extracellular fluid probably shifts during $+G_z$ acceleration (refs. 12 and 44) even though the time interval before this occurs is longer than the neural responses. However, it is very probable that the fluid shifts are not progressive (ref. 45) and are sufficiently small (circa 300 ml) that they are not detrimental to the cardiovascular dynamics (ref. 12). A sudden injection of a large amount of blood into the circulatory system usually increases the arterial, capillary, and venous pressures (ref. 43). Acceleration would cause the same effects as increasing the blood volume in the lower extremities and decreasing it in the upper extremities. However, the decrease of pressure in the upper half of the body dominates since nearly all pressosensors are in these upper regions. The blood volume is an extremely important regulator of blood pressure. It creates a pressure of its own in the circulatory system; with no blood flow, this pressure is called the mean circulatory filling pressure (ref. 43). Any decrease in the blood volume would lower the mean circulatory filling pressure and the cardiac output. When the blood volume drops to about 3000 ml, the mean circulatory filling pressure becomes essentially zero (ref. 43). Thus, the contribution of blood volume to blood pressure would be lost. Howard (ref. 22) observed a decrease in cardiac output of 32 percent at 2.0 G and 40 percent at 2.4 G in two supine subjects during $+G_z$ acceleration. In the experiments of Lindberg et al. (ref. 46), subjects seated upright exhibited a decrease in cardiac output of about 30 percent at 4.0 G. These results may be compared with the effects of 1.0 G (tilting the subject from the horizontal to the vertical position) which produced a decrease in cardiac output of about 25 percent (refs. 47 and 48).

The responses to $+G_z$ accelerations of relatively rapid onset (1 to 3 G/sec) are characterized by (1) a period of progressive deterioration - the pulse rate progressively increases, blood pressure declines, and there is a reduction in vision and consciousness; and (2) a period of compensation - the blood pressure rises, the pulse rate increases, and the vision often recovers (refs. 49-53). The period of progressive deterioration is usually alleviated in about 6 to 7 seconds by the cardiovascular compensatory reflexes (ref. 54). If the acceleration buildup is slow enough (circa 6.0 G/min), the cardiovascular reflexes have sufficient time to "keep up" with the hydrostatic forces induced by the acceleration and vision is not lost. Slow runs may be used to measure the reflex capacity of the subject (ref. 21). The acceleration profile in the present study was of the gradual onset type; we were assessing the full effects of the cardiovascular reflex mechanisms in our measurements of tolerance time. The specific nature of the reflex mechanism(s) during acceleration has not been defined, but their main function in $+G_z$ tolerance is to maintain the blood pressure. Blackout occurs when arterial pressure at eye level drops below about 20 mm Hg (refs. 3 and 42), and the cause of blackout has been attributed to retinal ischemic anoxia (ref. 52) and to a blockage of impulse propagation within the neural pathway (ref. 55). It is interesting that the pressoreceptors do not respond below about 30 mm Hg (ref. 43). This is approximately the pressure at the carotid sinus when the ocular pressure is 20 mm Hg.

Physiological reactions that help to maintain systemic blood pressure are increased stroke volume of the heart, tachycardia, arteriolar constriction (ref. 21), and venoconstriction (refs. 24 and 56). Since our subjects exerted

muscular force during centrifugation, our results must be interpreted as a combination centrifuge-muscular exercise problem.

Recent evidence has confirmed that exercise leads to dilatation of the resistance vessels (arterioles), an increase in the capillary filtration coefficient (ratio between precapillary and postcapillary resistances), and a distension of the capacitance (venules and veins) vessels. The dilatation of the latter was caused mainly by an increased pressure and not by an active venous dilatation (ref. 57). Warner et al. (ref. 58) have concluded that the fall in peripheral resistance plays an important role in bringing about the increase in heart rate and cardiac output that occurs in the early phases of exercise.

On the other hand, the reflex mechanisms during centrifugation without exercise would tend to constrict the arterioles and venules, thus decreasing the cardiac output - just the opposite of the exercise reaction. The beneficial effects of isometric muscular exercise on the capacity to maintain blood pressure during centrifugation must not only be centered around the mechanical restriction of blood flow, but must also aid venous return. These resistive effects probably outweigh the hypotensive effects caused by the drop in peripheral resistance which normally occurs with exercise, since muscular contraction is known to aid visual tolerance (ref. 35). Because the circulatory system reacts to the net effect of the two opposing influences, it is difficult to dissect the contribution of each separately.

The results of this study indicated a significant decrement in G tolerance (14.8 to 20.1 percent) of hypohydrated subjects, but essentially no difference in G tolerance between the acute and chronic hypohydration groups when they were allowed to exert muscular force during centrifugation. Tolerance time refers to time to black out during run 1. On the other hand, subsequent work has shown essentially no decrement in $+G_z$ tolerance in men hypohydrated up to 5 percent of their body weight who ride the centrifuge passively, without muscular effort (ref. 27). To our knowledge the only other study concerning this problem was conducted by Taliaferro et al. (ref. 30), who found a decrease in acceleration tolerance of 15 to 18 percent in subjects hypohydrated up to 3.5 percent in a hot room (51.7° C and 25-percent relative humidity) and riding passively. Thus, on the basis of the above studies, when heat was used as the method of hypohydration, a 15- to 20-percent tolerance decrement occurred regardless of whether the subjects rode passively or with muscular effort. However, if hypohydration was induced over a period of 5 days, there was no decrement in tolerance time in subjects who rode passively (ref. 27).

These results are in accord with the free circulating water (FCW) concept proposed by Ladell (ref. 59) and discussed by Greenleaf et al. (ref. 27). Briefly, the concept suggests that there is a reservoir of water in the body (about 2 liters or 2 to 3 percent of the body weight) over and above its critical needs that can be drawn upon in time of need. Until the store of FCW is depleted, the bodily functions could continue unimpaired. The site of the FCW has not been identified, but some of it is probably extracellular fluid. However, only 750 ml of the blood volume was lost in the H2a group. If the hypohydration were severe enough, the FCW might have been depleted faster than compensatory mechanisms could restore the lost fluid, thus causing the decrease in tolerance time. In longer term hypohydration (about 5 days) there would be

sufficient time for the compensatory mechanisms to come into play to maintain the volume of the FCW (ref. 27). Thus, performance could continue essentially unimpaired until the critical point was reached (fig. 11, 3-percent line) when the compensatory mechanisms would finally fail. The critical point would be variable depending upon, among other things, the rate and method of hypohydration and the physical condition of the subjects.

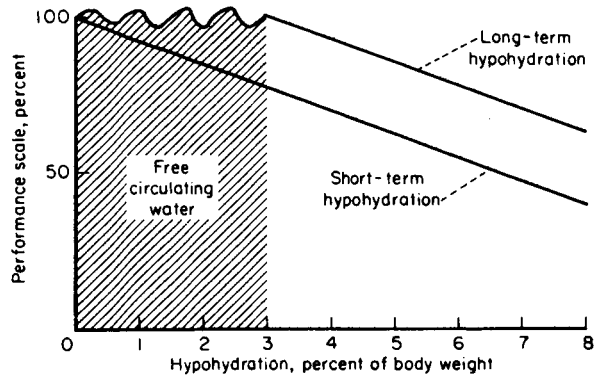

Figure 11.- Graphic concept of the relationship between hypohydration, performance, and free-circulating water.

In this experiment the 4-hour sauna bath (3.4-percent hypohydration) and the 48-hour water restriction period (3.8-percent hypohydrated) were approximately equivalent in reducing centrifuge tolerance. The blood volume losses, compared with their normohydration levels, were 750 and 400 ml, respectively. On the other hand, there was no clear relationship between body weight loss, blood volume, or plasma volume and tolerance time.

The chronic group was hyperglycemic as a result of the ingested sugar. However, there was only a slight decrease in the H2c fatigue effect. Centrifugation commenced about one hour following oral sugar ingestion, the time necessary for blood glucose to reach its maximum concentration (ref. 15). Clark et al. (ref. 12) found no significant effect on $+G_Z$ tolerance of either hyperglycemia (2 g sugar/kg) or insulin hypoglycemia (50 to 55 mg/100 ml blood glucose); however, hyperglycemia seemed to ameliorate the untoward effects of hyperventilation (dizziness, sweating, pallor, tachycardia, etc.) in conjunction with +3.4 G for 5 seconds.

CONCLUSIONS

Two groups of young male subjects were hypohydrated approximately 3.6 percent of their total body weight by means of a sauna bath (acute group) and a 48-hour water restriction period (chronic group) followed by four +3.7 G/min centrifugation runs which were held at 6.0 G until blackout occurred. Prior to centrifugation, each subject in the chronic hypohydration group was given 38 g of sugar in 100 ml of tap water.

In addition to glucose, serum K and urine osmolarity were the only biochemical measurements that differed significantly ($p < 0.05$) between the acute and chronic groups; the magnitude of the differences was not considered to be important physiologically. Some orthostatic intolerance was evident comparing reclining and standing pulse rates and blood pressures during hypohydration with the normohydration state.

Acute hypohydration reduced blackout tolerance 14.8 percent and chronic hypohydration 20.1 percent when compared with their respective normohydration tolerances. Thus, it may be concluded that:

1. Hypohydration reduces blackout tolerance 14 to 20 percent.

2. The decrease in tolerance is essentially independent of the method utilized and the time (4 hours or 48 hours) over which the water is lost.

3. Some orthostatic hypotension is associated with 3.6-percent hypohydration, and

4. No clear relationships could be demonstrated between body weight loss, total blood volume, plasma volume, and tolerance times.

Ames Research Center
National Aeronautics and Space Administration
Moffett Field, Calif., Apr. 5, 1966

REFERENCES

1. Von Diringshofen, H.: Die Wirkung von Fliehkräften auf den Blutkreislauf des im Flugzeug Sitzenden Menschen. Luftfahrtmedizin, vol. 6, 1942, pp. 152-165.

2. Livingston, P. C.: The Problem of "Black Out" in Aviation (Amaurosis Fugax). Brit. J. Surg., vol. 26, no. 104, April 1939, pp. 749-756.

3. Von Andina, F. (John F. Holman and Co., Inc., trans.): Ueber "Schwarzsehen" als Ausdruck von Blutdruckschwankimgen bei Sturzflugen. Schweizerische Medizinische Wochenschrift, vol. 18, no. 33, 1937, pp. 753-756.

4. Webb, M. G.: End Points for Acceleration Tolerances on the Centrifuge. Bio-Assay Techniques for Human Centrifuges and Physiological Effects of Acceleration, P. Bergeret, ed., Pergamon Press, N. Y., 1961, pp. 59-64.

5. Clark, William G.; and Jorgenson, Helen: Effect of Ingestion of Food and Fluid on Tolerance of Human Subjects to Positive Acceleration. Federation Proc., vol. 5, no. 1, March 1946, p. 17.

6. Clarke, Neville P.; Bondurant, Stuart; and Leverett, Sidney D.: Human Tolerance to Prolonged Forward and Backward Acceleration. J. Aviation Med., vol. 30, no. 1, Jan. 1959, pp. 1-21.

7. Dorman, Philip J.; and Lawton, Richard W.: Effect on G Tolerance of Partial Supination Combined With the Anti-G Suit. J. Aviation Med., vol. 27, no. 6, Dec. 1956, pp. 490-496.

8. Vykukal, Hubert C.; Gallant, Richard P.; and Stinnett, Glen W.: Description and Performance of an Interchangeable, Mobile Pilot Restraint System, Designed for Use in High Sustained Acceleration Force Fields. Aerospace Med., vol. 33, no. 3, March 1962, pp. 279-285.

9. Wood, Earl H.; Lindberg, Evan F.; Code, Charles F.; and Baldes, Edward J.: Effect of Partial Immersion in Water on Response of Healthy Men to Headward Acceleration. J. Appl. Physiol., vol. 18, no. 6, Nov. 1963, pp. 1171-1179.

10. Buhrlen, L.: Versuche Über die Bedeutung der Richtung beim Einwirken von Fliehkräften auf den menschlichen Körper. Luftfahrtmedizin, vol. 1, 1937, pp. 307-325.

11. Bjurstedt, Hilding; Hansson, Lars-Erik; and Ström, Gunnar: Electrocardiographic, Heart-Rate and Subjective Responses to Prolonged Gravitational Stress and Their Relation to Some Dimensional and Functional Parameters of the Circulatory System. Acta Physiol. Scand., vol. 47, no. 1, 1959, pp. 97-108.

12. Clark, William G.; Gardiner, I. D. R.; McIntyre, A. K.; and Jorgenson, Helen: Effect of Hyperglycemia and Insulin Hypoglycemia on Man's Tolerance to Positive Acceleration. Federation Proc., vol. 5, no. 1, March 1946, p. 17.

13. Hyde, Alvin S.; Cherniack, Neil S.; Lindberg, Evan F.; and Whately, Dorothy: Some Cardiorespiratory Responses of Flying and Non-Flying Personnel to Different Vectors of Acceleration With Correlation of These Responses to Other Variables. AMRL-TDR-62-151, Wright-Patterson Air Force Base, Ohio, Dec. 1962.

14. Meehan, John P.; and Jacobs, Howard I.: Relation of Several Physiological Parameters to Positive G Tolerance. WADC-TR 58-665, Wright-Patterson Air Force Base, Ohio, Jan. 1959.

15. Brent, H. P.; Carey, T. M.; Powell, T. J.; Scott, J. W.; Taylor, W. J. R.; and Franks, W. R.: Synergism Between Effects of Hyperventilation, Hypoglycemia and Positive Acceleration. Aerospace Med., vol. 31, no. 2, Jan. 1960, pp. 101-115.

16. Berry, Charles A.; Minners, Howard A.; McCutcheon, Ernest P.; and Pollard, Richard A.: Aeromedical Analysis. NASA SP-12, 1962, pp. 23-36.

17. Catterson, A. D.; McCutcheon, E. P.; Minners, E. P.; and Pollard, R. A.: Aeromedical Observations. NASA SP-45, 1963, pp. 299-326.

18. Minners, Howard A.; Douglas, William K.; Knoblock, Edward C.; Graybiel, Ashton; and Hawkins, Willard R.: Aeromedical Preparation and Results of Postflight Medical Examinations. Ch. 8 of Results of the First United States Manned Orbital Space Flight, Feb. 20, 1962, pp. 83-92.

19. Minners, Howard A.; White, Stanley C.; Douglas, William K.; Knoblock, Edward C.; and Graybiel, Ashton: Aeromedical Studies. A. Clinical Aeromedical Observations. NASA SP-6, 1962, pp. 43-53.

20. Johnson, Robert E.: Human Nutritional Requirements for Water in Long Space Flights. NASA SP-70, 1964, pp. 159-169.

21. Edelberg, Robert; Henry, James P.; Maciolek, John A.; Salzman, Edwin W.; and Zuidema, George D.: Comparison of Human Tolerance to Accelerations of Slow and Rapid Onset. J. Aviation Med., vol. 27, no. 6, Dec. 1956, pp. 482-489.

22. Howard, P.: Changes in the Cardiac Output During Positive Radial Acceleration. J. Physiol., vol. 147, April 17-18, 1959, pp. 49P-50P.

23. Henry, James P.; Gauer, Otto H.; Kety, Seymour S.; and Kramer, K.: Factors Maintaining Cerebral Circulation During Gravitational Stress. J. Clin. Invest., vol. 30, no. 3, March 1951, pp. 292-300.

24. Leverett, Sidney D.; Bondurant, Stuart; and Riley, Mitchell B.: Venous Constriction in Man During Exposure to Positive G Force. Federation Proc., vol. 16, no. 1, March 1957, p. 80.

25. Barr, Per-Olof: Pulmonary Gas Exchange in Man as Affected by Prolonged Gravitational Stress. Acta Physiol. Scand., vol. 58, supp. 207, 1963.

26. Black, D. A. K.: Dehydration. Lancet, vol. 1, March 10, 1945, pp. 298-300.

27. Greenleaf, J. E.; Matter, M., Jr.; Bosco, J. S.; Douglas, L. G.; and Averkin, E. G.: Effects of Hypohydration on Work Performance and Tolerance to $+G_z$ Acceleration in Man. Aerospace Med., vol. 37, no. 1, Jan. 1966, pp. 34-39.

28. Greenleaf, John E.; and Sargent, Frederick, II: Voluntary Dehydration in Man. J. Appl. Physiol., vol. 20, no. 4, July 1965, pp. 719-724.

29. Saltin, Bengt: Aerobic Work Capacity and Circulation at Exercise in Man With Special Reference to the Effect of Prolonged Exercise and/or Heat Exposure. Acta Physiol. Scand., vol. 62, supp. 230, 1964.

30. Taliaferro, E. H.; Wempen, R. R.; and White, W. J.: The Effect of Minimal Dehydration Upon Human Tolerance to Positive Acceleration. Paper 3114, Douglas Aircraft Co., Oct. 1964.

31. Dusterberry, John C.; and Barnett, Robert M: A Multipurpose Research Laboratory for Flight Simulation. Presented at the Aviation and Space, Hydraulic, and Gas Turbine Conference and Products Show, Los Angeles, Calif., March 3-7, 1963. ASME Paper 63-AHGT-87.

32. Howard, P.: Unconsciousness on the Human Centrifuge. Proc. Roy. Soc. Med., vol. 53, no. 1, Jan. 1960, pp. 99-100.

33. Brown, John L.: The Effect of Positive Acceleration on Visual Reaction Time. NADC-MA-5712, U. S. Naval Air Development Center, Johnsville, Pa., Aug. 12, 1957.

34. Howard, P.; and Byford, G. H.: Threshold Determination Techniques on the Human Centrifuge. FPRC Memo. 75, R. A. F. Institute of Aviation Medicine, Farnborough, England, Sept. 1956.

35. Raymond, S. A.; Greenleaf, John E.; and Matter, M., Jr.: Effects of Acute and Chronic Hypohydration on Tolerance to $+G_z$ Acceleration in Man. II. Impressions of Subjects. NASA TM X-1255, 1966.

36. Von Porat, Bertil T. D.: Blood Volume Determinations With the Evans Blue Dye Method. Acta Med. Scand., vol. 140, suppl. 256, 1951.

37. McGovern, Joseph J.; Jones, Alan Richardson; and Steinberg, Arthur G.: The Hematocrit of Capillary Blood. New Eng. J. Med., vol. 253, no. 8, Aug. 25, 1955, pp. 308-312.

38. Teller, Joseph D.: Direct, Quantitative, Colorimetric Determination of Serum or Plasma Glucose. Abstract 155, 130th Meeting Am. Chem. Soc., Sept. 1956, p. 69c.

39. Cotlove, Ernest; Trantham, Hillary V.; and Bowman, Robert L.: An Instrument and Method for Automatic, Rapid, Accurate, and Sensitive Titration of Chloride in Biologic Samples. J. Lab. Clin. Med., vol. 51, no. 3, March 1958, pp. 461-468.

40. Currens, James H.: A Comparison of the Blood Pressure in the Lying and Standing Positions: A Study of Five Hundred Men and Five Hundred Women. Am. Heart J., vol. 35, no. 4, April 1948, pp. 646-654.

41. Greenleaf, J. E.; Averkin, E. G.; and Sargent, Frederick, II: Water Consumption by Man in a Warm Environment: A Statistical Analysis. J. Appl. Physiol., vol. 21, no. 1, Jan. 1966, pp. 93-98.

42. Lambert, E. H.; and Wood, E. H.: Direct Determination of Man's Blood Pressure on the Human Centrifuge During Positive Acceleration. Federation Proc., vol. 5, no. 1, March 1946, p. 59.

43. Guyton, Arthur C.: Textbook of Medical Physiology. W. B. Saunders Co., Pa., 1961.

44. Fenichel, Richard L.; and Kydd, George H.: A Study of the Effects of Positive Acceleration Upon Erythrocyte Hydration in Human Subjects. Aerospace Med., vol. 33, no. 7, July 1962, pp. 862-865.

45. Miller, Hugh; Riley, Mitchell B.; Bondurant, Stuart; and Hiatt, Edwin P.: The Duration of Tolerance to Positive Acceleration. J. Aviation Med., vol. 30, no. 5, May 1959, pp. 360-366.

46. Lindberg, Evan F.; Sutterer, William F.; Marshall, Hiram W.; Headley, Robert N.; and Wood, Earl H.: Measurement of Cardiac Output During Headward Acceleration Using the Dye-Dilution Technique. Aerospace Med., vol. 31, no. 10, Oct. 1960, pp. 817-834.

47. McMichael, John; and Sharpey-Schafer, E. P.: Cardiac Output in Man by a Direct Fick Method. Effects of Posture, Venous Pressure Change, Atropine, and Adrenaline. Brit. Heart J., vol. 6, no. 1, Jan. 1944, pp. 33-40.

48. Stead, E. A., Jr.; Warren, J. V.; Merrill, A. J.; and Brannon, E. S.: The Cardiac Output in Male Subjects as Measured by the Technique of Right Atrial Catheterization, Normal Values With Observations on the Effect of Anxiety and Tilting. J. Clin. Invest., vol. 24, May 1945, pp. 326-331.

49. Code, C. F.; Wood, E. H.; Sturm, R. E.; Lambert, E. H.; and Baldes, E. J.: The Sequence of Physiologic Events in Man During Exposure to Positive Acceleration. Federation Proc., vol. 4, no. 1, March 1945, pp. 14-15.

50. Maison, George L.; Maaske, C. A.; and Martin, E. E.: Sensory Effects of Centrifugal Force on Man When Seated. Federation Proc., vol. 4, no. 1, March 1945, pp. 48-49.

51. Stewart, W. K.: Investigations on Centrifugal Force. J. Physiol., vol. 104, June 29, 1945, pp. 7P-8P.

52. Stewart, W. K.: Some Observations on the Effect of Centrifugal Force in Man. J. Neurol. Neurosurg. Psychiat., vol. 8, nos. 1 and 2, Jan.-April 1945, pp. 24-33.

53. Duane, T. D.: Observations on the Fundus Oculi During Black-Out. A.M.A. Arch. Opthal., vol. 51, no. 3, March 1954, pp. 343-355.

54. Maciolek, John A.: Circulatory Reflex Activity as a G-Protective Device. WADC TN-WCRD 55-1, Wright-Patterson Air Force Base, Ohio, Jan. 1955.

55. Noell, Werner K.: Site of Asphyxial Block in Mammalian Retinae. J. Appl. Physiol., vol. 3, no. 8, Feb. 1951, pp. 489-500.

56. Salzman, Edwin W.; and Leverett, Sidney D.: Orthostatic Venoconstriction Studied by a Miniature Balloon Technique. Federation Proc., vol. 15, no. 1, March 1956, pp. 160-161.

57. Kjellmer, Ingemar: The Effect of Exercise on the Vascular Bed of Skeletal Muscle. Acta Physiol. Scand., vol. 62, nos. 1-2, Sept.-Oct. 1964, pp. 18-30.

58. Warner, Homer R.; Topham, W. Sanford; and Nicholes, Karl K.: The Role of Peripheral Resistance in Controlling Cardiac Output During Exercise. Ann. N. Y. Acad. Sci., vol. 115, Art. 2, July 31, 1964, pp. 669-679.

59. Ladell, W. S. S.: The Effects of Water and Salt Intake Upon the Performance of Men Working in Hot and Humid Environments. J. Physiol., vol. 127, no. 1, Jan. 1955, pp. 11-46.

TABLE I.- PULSE RATES AND BLOOD PRESSURES

Subject	Pulse rates			Blood pressures				
	Lying	Standing	Standing minus lying	Lying	Standing	Standing minus lying	Standing 2 minutes	Standing minus 2 minutes standing
Normohydration (N1)								
MM	-	-	-	154/82	-	-	-	-
HV	76	-	-	120/58	110/84	-10/26	-	-
WL	60	-	-	110/58	120/76	10/18	108/70	12/6
RP	57	-	-	130/68	130/86	0/18	130/80	0/6
JM	75	066	-09	112/68	116/82	4/14	110/84	6/-2
DD	51	069	18	128/80	142/102	14/22	130/102	12/0
JB	66	084	18	118/92	112/100	-6/8	110/98	2/2
JL	64	072	08	120/74	132/84	12/10	126/88	6/-4
MP	66	078	12	128/92	142/110	14/18	132/116	10/-6
DL	72	072	00	164/88	204/110	40/22	174/120	30/-10
CO	68	072	04	118/70	122/94	4/24	120/94	2/0
FK	66	075	09	124/70	134/78	10/8	126/80	8/-2
JG	66			120/68	-	-	-	-
$\bar{X}$	65.6	074	7.5	126.6/74.5	133.1/91.4	8.4/17.1	126.6/93.2	8.9/-1.0
±SE	2.07	1.97	2.37	4.34/3.2	7.84/3.73	3.97/1.91	6.0/5.1	2.70/1.58
n	12	8	8	13	11	11	10	10
Hypohydration (H2a)								
MM	69	100	31	130/82	110/86	-20/4	108/88	2/-2
HV	75	120	45	118/0	90/78	-28/78	100/74	-10/4
WL	74	093	19	106/68	86/80	-20/12	98/74	-12/6
RP	48	063	15	118/68	106/78	-12/10	112/88	-6/-10
JM	63	093	30	118/94	144/108	26/14	130/98	14/10
$\bar{X}$	65.800	93.800	28.000	118.00/62.400	107.20/86.00	-10.800/23.600	109.60/84.400	-2.400/1.600
±SE	4.93	9.15	5.25	3.80/16.34	10.27/5.69	9.54/13.70	5.71/4.62	4.75/3.49
n	5	5	5	5	5	5	5	5
Hypohydration (H2c)								
DD	58	096	38	128/84	128/100	0/16	134/100	-6/0
JB	75	082	07	118/74	110/80	-8/6	112/80	-2/10
JL	63	069	06	114/78	126/78	12/0	106/84	20/-6
MP	72	087	15	122/84	116/100	-6/16	118/90	-2/10
DL	62	078	16	180/74	164/90	-16/16	170/110	-6/-20
CO	51	084	33	108/80	122/108	14/28	128/100	-8/8
FK	78	126	48	134/90	148/112	14/22	128/98	20/14
JG	60	084	24	110/80	98/80	-12/0	112/80	-14/0
$\bar{X}$	64.875	88.250	23.375	126.75/80.500	126.50/93.500	-0.250/13.000	126.00/92.750	0.250/0.750
±SE	3.28	6.03	5.36	8.22/1.92	7.42/4.73	4.30/3.58	7.15/3.87	4.51/3.76
n	8	8	8	8	8	8	8	8
Hypohydration (H2a and H2c)								
$\bar{X}$	65.231	90.385	25.154	123.38/73.538	119.08/90.615	-4.308/17.077	119.69/89.538	-0.769/1.0769
±SE	2.64	4.94	3.78	5.26/6.48	6.38/3.65	4.53/5.56	5.28/3.08	3.21/2.58
n	13	13	13	13	13	13	13	13
Normohydration (N3a)								
MM	60	072	12	128/92	128/88	0/-4	124/102	4/-14
HV	81	087	06	124/50	130/76	6/26	130/84	0/-8
WL	54	068	14	100/66	120/78	20/12	104/78	16/0
RP	50	054	04	124/76	136/88	12/12	128/80	8/8
JM	60	078	18	122/94	132/112	10/18	128/110	4/2
$\bar{X}$	61.000	71.800	10.800	119.60/75.600	129.20/88.400	9.600/12.800	122.800/90.800	6.400/-2.400
±SE	5.35	5.48	2.58	5.00/8.23	2.65/6.40	3.31/4.92	4.80/6.41	2.71/3.87
n	5	5	5	5	5	5	5	5
Normohydrstion (N3c)								
DD	44	066	22	122/84	132/90	10/6	128/98	4/-8
JB	58	058	00	108/78	128/92	20/14	124/92	4/0
JL	62	060	-02	124/76	126/80	2/4	120/84	6/-4
MP	64	074	10	134/78	138/90	4/12	126/96	12/-6
DL	74	078	04	168/76	160/90	-8/14	154/100	6/-10
CO	52	068	16	110/68	110/86	0/18	108/80	2/6
FK	60	075	15	122/88	132/96	10/8	128/108	4/-12
JG	66	084	18	116/68	114/78	-2/10	128/100	-14/-22
$\bar{X}$	60.000	70.625	10.625	125.50/77.000	130.00/87.750	4.500/10.750	127.00/94.750	3.000/-7.000
±SE	3.21	3.06	2.99	6.74/2.45	5.42/2.15	3.06/1.64	4.54/3.23	2.64/2.95
n	8	8	8	8	8	8	8	8
Normohydration (N3a and N3c)								
$\bar{X}$	60.385	71.077	10.692	123.23/76.462	129.69/88.000	6.462/11.538	125.38/93.231	4.308/-5.2308
±SE	2.72	2.69	2.02	4.49/3.30	3.39/2.63	2.30/2.04	3.27/3.05	1.92/2.34
n	13	13	13	13	13	13	13	13

TABLE II.- BLOOD VARIABLES

Subject	Serum glucose, mg/100 ml	Evans blue space, ml	Red cell volume, ml	Blood volume, ml	Hematocrit, vol %	Serum Na, mEq/l	Serum K, mEq/l	Serum Cl, mEq/l	Serum osmolarity, mOsm/l
Normohydration (N1)									
MM	085	3181	2293	5473	43.7	142.9	4.6	102.7	278.0
HV	092	3341	2399	5740	43.5	140.4	4.0	99.7	276.0
WL	083	3612	2398	6010	41.6	140.0	4.9	101.9	278.0
RP	101	3322	2178	5500	41.4	144.0	4.4	102.4	274.0
JM	090	2984	2143	5127	43.5	143.5	4.0	103.3	284.0
DD	097	5319	4013	9332	44.8	143.6	3.9	101.2	282.0
JB	093	3215	2328	5543	43.7	141.4	4.1	105.0	310.0
JL	085	2947	2334	5281	46.0	143.2	4.6	099.4	294.0
MP	090	3445	2120	5565	39.7	144.5	4.2	101.6	294.0
DL	094	3301	2756	6057	47.4	145.0	4.5	100.8	302.0
CO	076	3858	2456	6314	40.5	144.4	3.8	099.8	286.0
FK	091	3633	2707	6340	44.5	146.0	4.6	103.8	290.0
JG	083	3218	2359	5577	44.1	143.8	4.7	101.2	276.0
$\bar{X}$	089	3490	2499	5989	43.4	143.3	4.3	101.8	286.0
±SE	1.86	168	188.9	297	0.599	0.49	0.1	0.46	3.08
n	13	13	13	13	13	13	13	13	13
Hypohydration (H2a)									
MM	090	3136	2770	5906	48.9	146.0	4.6	104.6	280.0
HV	091	2856	2327	5183	46.8	148.8	5.1	099.4	292.9
WL	086	3231	2398	5629	44.4	146.5	4.9	101.1	296.8
RP	086	3657	2338	5995	40.6	149.5	4.3	109.0	302.0
JM	144	3065	2275	5340	44.4	147.4	5.0	102.8	292.9
$\bar{X}$	99.40	3189.0	2421.6	5610.6	45.020	147.64	4.7800	103.38	292.92
±SE	11.20	132.2	89.3	156.8	1.39	0.67	0.15	1.65	3.64
n	5	5	5	5	5	5	5	5	5
Hypohydration (H2c)									
DD	126	4046	3559	7605	48.8	145.0	4.4	096.4	294.0
JB	147	3468	2584	6052	44.5	148.0	4.4	101.6	293.5
JL	160	2894	2515	5409	48.4	145.5	4.9	101.4	300.0
MP	143	3284	2117	5401	40.8	145.2	4.6	100.4	302.0
DL	113	3164	2828	5992	49.2	149.8	4.6	101.4	286.0
CO	099	3322	2496	5818	44.7	147.0	4.6	096.4	294.0
FK	135	3209	2778	5987	48.3	147.7	4.8	101.4	294.0
JG	143	2994	2490	5484	47.3	146.0	4.4	103.8	300.0
$\bar{X}$	133.25	3297.6	2670.9	5968.5	46.500	146.77	4.5875	100.35	295.44
±SE	6.99	124.7	148.0	252.8	1.03	0.59	0.07	0.93	1.81
n	8	8	8	8	8	8	8	8	8
Hypohydration (H2a and H2c)									
$\bar{X}$	120.23	3255.8	2575.0	5830.8	45.931	147.11	4.6615	101.52	294.47
±SE	7.50	89.8	100.6	169.2	0.82	0.44	0.07	0.92	1.73
n	13	13	13	13	13	13	13	13	13
Normohydration (N3a)									
MM	083	3555	2771	6326	45.6	142.7	5.6	100.9	287.5
HV	116	3924	2900	6824	44.3	144.5	4.1	102.0	290.0
WL	074	3258	2612	5870	46.4	146.6	5.2	103.0	292.0
RP	092	3840	2592	6432	42.0	145.5	4.2	104.5	290.0
JM	082	3693	2620	6313	43.2	152.0	4.7	098.4	272.0
$\bar{X}$	89.40	3654.0	2699.0	6353.0	44.300	146.26	4.7600	101.76	286.30
±SE	7.24	117.4	59.5	152.3	0.79	1.57	0.29	1.03	3.64
n	5	5	5	5	5	5	5	5	5
Normohydration (N3c)									
DD	083	4477	2575	8052	46.2	145.5	3.9	099.4	284.0
JB	093	3793	2497	6290	41.4	142.4	3.6	100.9	298.0
JL	084	2862	2560	5422	49.2	144.5	4.2	103.0	290.1
MP	085	3765	2230	5995	38.7	144.7	4.3	099.9	290.1
DL	107	3422	2974	6396	48.4	145.0	4.3	100.4	280.0
CO	082	4053	2548	6601	40.2	143.8	4.0	102.2	280.0
FK	079	3615	2727	6342	44.8	144.0	4.2	102.5	286.0
JG	118	3400	2382	5782	42.9	147.4	4.3	103.5	293.5
$\bar{X}$	91.38	3673.4	2686.6	6360.0	43.975	144.66	4.1000	101.47	287.71
±SE	4.93	169.7	149.2	276.3	1.35	0.51	0.09	0.54	2.26
n	8	8	8	8	8	8	8	8	8
Normohydration (N3a and N3c)									
$\bar{X}$	90.62	3665.9	2691.4	6357.3	44.100	145.28	4.3538	101.58	287.17
±SE	3.94	110.0	91.9	174.3	0.86	0.68	0.15	0.49	1.89
n	13	13	13	13	13	13	13	13	13

TABLE III.- URINE VARIABLES

Subject	Urine volume, ml	Urine Na, mEq/l	Urine K, mEq/l	Urine Cl, mEq/l	Urine osmolarity, mOsm/l
Normohydration (N1)					
MM	-	050	016	055.7	0274.0
HV	077	120	084	190.8	0786.0
WL	249	176	042	173.3	0740.0
RP	025	130	052	144.2	1000.0
JM	128	110	036	116.3	1058.0
DD	170	112	050	118.9	0484.0
JB	030	156	094	192.3	1074.0
JL	005	122	060	127.2	0932.0
MP	012	190	028	197.9	0834.0
DL	030	188	074	228.3	1180.0
CO	030	100	112	097.3	1148.0
FK	210	252	100	326.1	0966.0
JG	210	156	091	200.4	0896.0
$\bar{X}$	098	143	64	166.8	875
±SE	26.06	14.15	8.38	19.05	68.65
n	12	13	13	13	13
Hypohydration (H2a)					
MM	038	066	068	141.2	0852.0
HV	007	020	160	077.7	0764.0
WL	025	088	152	098.2	1162.0
RP	040	194	090	216.8	0896.0
JM	074	052	080	080.2	0918.0
$\bar{X}$	36.80	84.000	110.00	122.820	918.40
±SE	11.01	29.63	19.14	26.10	66.37
n	5	5	5	5	5
Hypohydration (H2c)					
DD	260	052	092	081.7	0936.0
JB	065	048	164	107.5	1112.0
JL	030	098	056	092.1	0820.0
MP	110	038	120	076.7	0676.0
DL	125	080	140	147.8	1300.0
CO	152	056	148	113.1	0988.0
FK	142	026	128	064.8	1012.0
JG	025	062	140	115.1	1154.0
$\bar{X}$	113.63	57.500	123.50	99.850	999.75
±SE	27.08	8.09	12.22	9.37	69.18
n	8	8	8	8	8
Hypohydration (H2a and H2c)					
$\bar{X}$	84.077	67.692	118.31	108.68	968.46
±SE	19.88	12.24	10.21	11.37	49.12
n	13	13	13	13	13

TABLE III.- URINE VARIABLES - Concluded

Subject	Urine volume, ml	Urine Na, mEq/l	Urine K, mEq/l	Urine Cl, mEq/l	Urine osmolarity, mOsm/l
Normohydration (N3a)					
MM	080	066	061	070.9	0408.0
HV	160	124	065	171.6	0536.0
WL	125	036	069	074.2	0586.0
RP	006	240	105	258.8	0984.0
JM	150	088	027	102.8	0476.0
$\bar{X}$	104.20	110.800	65.40	135.660	598.00
±SE	28.17	35.35	12.40	35.71	100.99
n	5	5	5	5	5
Normohydration (N3c)					
DD	350	168	096	181.8	0714.0
JB	028	198	055	213.4	1014.0
JL	050	164	051	174.4	0984.0
MP	060	134	104	131.3	0804.0
DL	088	264	076	262.4	0946.0
CO	028	152	064	211.4	1048.0
FK	052	188	112	268.3	0708.0
JG	030	196	055	223.6	0994.0
$\bar{X}$	85.75	183.000	76.63	208.325	895.25
±SE	38.43	13.97	8.58	16.13	47.53
n	8	8	8	8	8
Normohydration (N3a and N3c)					
$\bar{X}$	92.846	155.231	72.31	180.38	780.92
±SE	25.27	18.25	6.98	19.00	62.14
n	13	13	13	13	13

TABLE IV.- CENTRIFUGE TOLERANCE TIMES AND HEART RATES

	Run 1			Run 2			Run 3			Run 4		
Subject	Seconds	Rates begin/end	Δ PR	Seconds	Rates begin/end	Δ PR	Seconds	Rates begin/end	Δ PR	Seconds	Rates begin/end	Δ PR
Normohydration (N1)												
MM	094.0	132/174		096.0	144/174		095.0	132/174		095.0	132/168	
HV	089.0	126/180		092.5	108/192		-	-		-	-	
WL	101.0	72/156		101.0	78/156		093.0	78/150		-	-	
RP	091.0	84/162		067.0	114/144		061.0	102/108		050.5	78/102	
JM	079.5	132/150		083.5	120/150		088.0	120/162		091.0	126/174	
DD	075.5	66/132		070.0	90/120		-	-		-	-	
JB	082.5	144/174		083.0	126/156		083.0	108/174		082.0	126/168	
JL	059.5	78/102		066.5	108/102		055.5	96/120		054.5	78/108	
MP	081.0	90/150		081.0	114/156		083.0	120/168		081.0	102/188	
DL	086.5	96/144		085.5	96/138		-	-		085.5	90/138	
CO	075.5	108/108		073.0	96/120		071.5	84/102		065.0	96/108	
FK	084.0	96/144		078.5	96/126		084.5	96/138		084.0	84/144	
JG	098.5	84/156		090.0	108/156		072.5	108/126		-	-	
$\bar{X}$	84.4	101/149		82.1	108/145		78.7	104/142		76.5	101/144	
±SE	3.05	7.07/6.56		3.05	4.73/6.73		3.8	5.20/8.63		5.32	7.36/10.79	
n	13	13		13	13		10	10		9	9	
Hypohydration (H2a)												
MM	167.0	114/186	72	113.0	114/180	66	102.0	144/180	36	098.5	126/180	54
HV	046.0	120/150	30	061.0	108/180	72	064.5	114/222	108	064.0	132/264	132
WL	085.0	96/168	72	080.5	108/162	54	072.0	102/144	42	063.5	102/144	42
RP	070.0	102/186	84	066.5	90/150	60	068.5	90/150	60	067.5	96/150	54
JM	079.5	132/168	36	079.5	114/162	48	082.5	114/168	54	072.5	114/168	54
$\bar{X}$	89.50	113/172	59	80.10	107/167	60	77.90	113/173	60	73.20	114/181	67
±SE	20.49	6.40/6.74	10.8	9.04	4.41/5.82	4.2	6.73	8.98/13.86	12.7	6.52	6.84/21.67	16.4
n	5	5	5	5	5	5	5	5	5	5	5	5
Hypohydration (H2c)												
DD	088.5	78/150	72	085.5	102/144	42	082.5	108/144	36	088.5	102/144	42
JB	085.0	- /168	-	084.5	120/180	60	078.0	114/162	48	078.0	96/168	72
JL	069.5	96/138	42	079.5	96/144	48	080.5	90/144	54	074.5	90/132	42
MP	084.0	102/186	84	085.0	138/186	48	085.0	132/186	54	084.0	138/186	42
DL	087.0	90/180	90	100.5	126/186	60	094.0	126/192	66	100.5	126/186	60
CO	074.5	96/138	42	063.0	84/114	30	064.5	84/120	36	064.0	84/126	42
FK	095.5	108/192	84	077.7	138/186	50	083.5	138/180	42	081.0	144/180	36
JG	079.0	102/162	60	079.0	120/168	48	060.5	108/150	42	072.0	108/162	54
$\bar{X}$	082.9	96/164	68	080.7	116/164	48	78.6	112/160	47	80.31	111/160	49
±SE	2.95	3.70/7.49	8.0	3.72	8.45/9.47	3.4	3.89	6.32/8.78	3.7	3.92	7.94/8.47	4.3
n	8	7 8	7	8	8	8	8	8	8	8	8	8
Normohydration (N3a)												
MM	126.0	96/162	66	116.0	90/150	60	100.5	96/150	54	085.0	96/126	30
HV	112.5	102/186	84	099.5	138/180	42	090.0	132/186	54	087.0	132/180	48
WL	093.0	66/150	84	094.0	60/ -	-	079.5	- /132	-	079.5	66/156	90
RP	098.5	66/150	84	100.5	72/156	84	096.5	102/156	54	101.0	90/150	60
JM	096.5	114/174	60	087.5	84/168	84	084.0	108/156	48	087.5	114/168	54
$\bar{X}$	105.3	89/164	76	99.5	89/164	68	90.1	110/156	52	88.0	100/156	56
±SE	6.09	9.75/7.00	5.2	4.73	13.34/9.28	10.2	3.86	7.90/8.69	1.5	3.55	11.16/9.10	9.8
n	5	5	5	5	5 4	4	5	4 5	4	5	5	5
Normohydration (N3c)												
DD	091.5	66/144	78	083.5	96/144	48	078.5	90/138	48	074.5	78/138	60
JB	107.0	108/168	60	098.5	114/174	60	095.0	102/168	66	099.0	90/162	72
JL	130.5	78/150	72	084.5	102/132	30	085.0	96/126	30	091.0	96/138	42
MP	106.5	102/180	78	105.0	126/174	48	107.0	132/186	54	110.0	132/180	48
DL	121.5	90/186	96	105.0	126/186	60	103.5	126/186	60	108.5	132/186	54
CO	059.0	72/120	48	088.0	72/144	72	075.0	66/138	72	084.0	84/144	60
FK	095.5	102/186	84	090.5	126/168	42	086.5	120/168	48	082.5	120/162	42
JG	117.5	84/162	78	087.0	90/162	72	075.5	102/144	42	073.5	90/150	60
$\bar{X}$	103.6	88/162	74	92.8	106/160	54	88.2	104/157	53	90.4	103/158	55
±SE	7.86	5.43/6.68	5.2	3.13	7.06/6.59	5.2	4.33	9.60/8.21	4.8	5.05	7.73/6.49	3.7
n	8	8	8	8	8	8	8	8	8	8	8	8
Normohydration (N3a and N3c)												
$\bar{X}$	104.3	88/163		95.3	100/162		89.0	106/156		89.5	102/157	
±SE	5.18	4.74/5.54		2.7	6.84/4.76		2.98	5.54/5.82		3.3	6.13/5.08	
n	13	13		13	13 12		13	12 13		13	13	

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES

(a) Raw data summary
(Mean values ±SE)

		Hypohydration	Normohydration	$\bar{X}$
Lying pulse rate, beats/min	Chronic	64.9 (3.28)	60.0 (3.21)	62.4
	Acute	65.8 (4.93)	61.0 (5.35)	63.4
	$\bar{X}$	65.2	60.4	
Standing pulse rate, beats/min	Chronic	88.2 (6.03)	70.4 (3.06)	79.3
	Acute	93.8 (9.15)	71.8 (5.48)	82.8
	$\bar{X}$	90.4	70.9	
Standing minus lying pulse rate	Chronic	23.4 (5.36)	10.4 (2.99)	16.9
	Acute	28.0 (5.25)	10.8 (2.58)	19.4
	$\bar{X}$	25.2	10.5	
Lying systolic blood pressure	Chronic	126.8 (8.22)	125.5 (6.74)	126.2
	Acute	118.0 (3.80)	119.6 (5.00)	118.8
	$\bar{X}$	123.4	123.2	
Standing systolic blood pressure	Chronic	126.5 (7.42)	130.0 (5.42)	128.2
	Acute	107.2 (10.27)	129.2 (2.65)	118.2
	$\bar{X}$	119.1	129.7	
Standing minus lying systolic blood pressure	Chronic	-0.3 (4.30)	4.5 (3.06)	2.1
	Acute	-10.8 (9.54)	9.6 (3.31)	-0.6
	$\bar{X}$	-4.3	6.5	
Lying diastolic blood pressure	Chronic	80.5 (1.92)	77.0 (2.45)	78.8
	Acute	78.0 (16.34)	82.0 (8.23)	80.0
	$\bar{X}$	79.7	78.7	
Standing diastolic blood pressure	Chronic	93.5 (4.73)	87.8 (2.15)	90.6
	Acute	86.0 (5.69)	88.4 (6.40)	87.2
	$\bar{X}$	90.6	88.0	
Standing minus lying diastolic blood pressure	Chronic	13.0 (3.58)	10.8 (1.64)	11.9
	Acute	23.6 (13.70)	12.8 (4.92)	18.2
	$\bar{X}$	17.1	11.5	
2-min standing systolic blood pressure	Chronic	126.0 (7.15)	127.0 (4.54)	126.5
	Acute	109.6 (5.71)	122.8 (4.80)	116.2
	$\bar{X}$	119.7	125.4	
Δ standing systolic blood pressure	Chronic	-0.3 (4.51)	3.0 (2.64)	1.6
	Acute	-2.4 (4.75)	6.4 (2.71)	2.0
	$\bar{X}$	-0.8	4.3	

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES - Continued

(a) Raw data summary - Continued
(Mean values ±SE)

		Hypohydration	Normohydration	$\bar{X}$
2-min standing diastolic blood pressure	Chronic	92.8 (3.87)	94.8 (3.23)	93.8
	Acute	84.4 (4.62)	90.8 (6.41)	87.6
	$\bar{X}$	89.5	93.2	
Δ standing diastolic blood pressure	Chronic	0.8 (3.76)	-7.0 (2.95)	-3.1
	Acute	1.6 (3.49)	-2.4 (3.87)	-0.4
	$\bar{X}$	1.1	-5.2	
Serum glucose, mg/100 ml	Chronic	133.2 (6.99)	91.4 (4.93)	112.3
	Acute	99.4 (11.20)	89.4 (7.24)	94.4
	$\bar{X}$	120.2	90.6	
Evans blue space, ml	Chronic	3297.6 (124.7)	3673.4 (169.7)	3485.5
	Acute	3189.0 (132.2)	3654.0 (117.4)	3421.5
	$\bar{X}$	3255.8	3665.9	
Red cell volume, ml	Chronic	2670.9 (148.0)	2686.6 (149.2)	2678.8
	Acute	2421.6 (89.3)	2699.0 (59.5)	2560.3
	$\bar{X}$	2575.0	2691.4	
Blood volume, ml	Chronic	5968.5 (252.8)	6360.0 (276.3)	6164.2
	Acute	5610.6 (156.8)	6353.0 (152.3)	5981.8
	$\bar{X}$	5830.8	6357.3	
Hematocrit, vol %	Chronic	46.5 (1.03)	44.0 (1.35)	45.2
	Acute	45.0 (1.39)	44.3 (0.79)	44.6
	$\bar{X}$	45.9	44.1	
Serum Na, mEq/l	Chronic	146.8 (0.59)	144.7 (0.51)	145.8
	Acute	147.6 (0.67)	146.3 (1.57)	147.0
	$\bar{X}$	147.1	145.3	
Serum K, mEq/l	Chronic	4.6 (0.07)	4.1 (0.09)	4.4
	Acute	4.8 (0.15)	4.8 (0.29)	4.8
	$\bar{X}$	4.7	4.4	
Serum Cl, mEq/l	Chronic	100.4 (0.93)	101.5 (0.54)	100.9
	Acute	103.4 (1.65)	101.8 (1.03)	102.6
	$\bar{X}$	101.5	101.6	
Serum osmolarity, mOsm/l	Chronic	295.4 (1.81)	287.7 (2.26)	291.6
	Acute	292.9 (3.64)	286.3 (3.64)	289.6
	$\bar{X}$	294.5	287.2	

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES - Continued

(a) Raw data summary - Concluded
(Mean values ±SE)

		Hypohydration	Normohydration	$\bar{X}$
Urine volume, ml	Chronic	113.6 (27.08)	85.8 (38.43)	99.7
	Acute	36.8 (11.01)	104.2 (28.17)	70.5
	$\bar{X}$	84.1	92.8	
Urine Na, mEq/l	Chronic	57.5 (8.09)	183.0 (13.97)	120.2
	Acute	84.0 (29.63)	110.8 (35.35)	97.4
	$\bar{X}$	67.7	155.2	
Urine K, mEq/l	Chronic	123.5 (12.22)	76.6 (8.58)	100.1
	Acute	110.0 (19.14)	65.4 (12.40)	87.7
	$\bar{X}$	118.3	72.3	
Urine Cl, mEq/l	Chronic	99.8 (9.37)	208.3 (16.13)	154.1
	Acute	122.8 (26.10)	135.7 (35.71)	129.2
	$\bar{X}$	108.7	180.4	
Urine osmolarity, mOsm/l	Chronic	999.8 (69.18)	895.2 (47.53)	947.5
	Acute	918.4 (66.37)	598.0 (100.99)	758.2
	$\bar{X}$	968.5	780.9	
Tolerance time	Chronic	80.9 (1.73)	93.8 (8.09)	87.4
	Acute	80.2 (5.57)	95.7 (2.14)	88.0
	$\bar{X}$	80.5	95.0	

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES - Continued

(b) Analysis of variance data

Source	Degree of freedom	Sum of squares	Mean square	F	p
Lying pulse rate					
Method	1	5.6	5.6	0.04	n.s.
Error (a)	11	1595.8	145.1	2.49	n.s.
State	1	152.6	152.6	2.62	n.s.
M × S	1	0.1	0.1	0.002	n.s.
Error (b)	11	639.8	58.2		
Standing pulse rate					
Method	1	74.9	74.9	0.22	n.s.
Error (a)	11	3671.5	333.8	3.05	< 0.05
State	1	2461.9	2461.9	22.46	< 0.001
M × S	1	26.1	26.1	0.24	n.s.
Error (b)	11	1205.5	109.6		
Standing minus lying pulse rate					
Method	1	39.2	39.2	0.19	n.s.
Error (a)	11	2244.2	204.0	3.76	< 0.025
State	1	1388.5	1388.5	25.62	< 0.001
M × S	1	27.1	27.1	0.50	n.s.
Error (b)	11	596.4	54.2		
Lying systolic blood pressure					
Method	1	330.1	330.1	0.54	n.s.
Error (a)	11	6707.4	609.8	16.57	< 0.001
State	1	0.1	0.1	0.003	n.s.
M × S	1	12.6	12.6	0.34	n.s.
Error (b)	11	405.3	36.8		
Standing systolic blood pressure					
Method	1	621.6	621.6	1.26	n.s.
Error (a)	11	5432.6	493.9	3.50	< 0.025
State	1	732.5	732.5	5.20	< 0.05
M × S	1	526.2	526.2	3.73	n.s.
Error (b)	11	1551.0	141.0		

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES - Continued

(b) Analysis of variance data - Continued

Source	Degree of freedom	Sum of squares	Mean square	F	p
Standing minus lying systolic blood pressure					
Method	1	45.6	45.6	0.25	n.s.
Error (a)	11	2008.2	182.6	1.26	n.s.
State	1	753.8	753.8	5.20	< 0.05
M × S	1	376.9	376.9	3.43	n.s.
Error (b)	11	1593.3	144.9		
Lying diastolic blood pressure					
Method	1	8.3	8.3	0.06	n.s.
Error (a)	10	1371.0	137.1	7.66	< 0.005
State	1	6.0	6.0	0.34	n.s.
M × S	1	75.0	75.0	4.19	n.s.
Error (b)	10	179.0	17.9		
Standing diastolic blood pressure					
Method	1	72.1	72.1	0.31	n.s.
Error (a)	11	2517.4	228.9	5.44	< 0.005
State	1	44.4	44.4	1.05	n.s.
M × S	1	102.3	102.3	2.43	n.s.
Error (b)	11	463.3	42.1		
Standing minus lying diastolic blood pressure					
Method	1	246.1	246.1	0.73	n.s.
Error (a)	11	3731.4	339.2	2.70	n.s.
State	1	199.3	199.3	1.59	n.s.
M × S	1	112.6	112.6	0.90	n.s.
Error (b)	11	1380.1	125.5		
2-min standing systolic blood pressure					
Method	1	652.9	652.9	1.72	n.s.
Error (a)	11	4165.6	378.6	4.33	< 0.025
State	1	210.6	210.6	2.41	n.s.
M × S	1	228.9	228.9	2.62	n.s.
Error (b)	11	962.4	87.5		

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES - Continued

(b) Analysis of variance data - Continued

Source	Degree of freedom	Sum of squares	Mean square	F	p
Δ standing systolic blood pressure					
Method	1	0.9	0.9	0.01	n.s.
Error (a)	11	1,247.8	113.4	1.41	n.s.
State	1	167.5	167.5	2.09	n.s.
M × S	1	56.4	56.4	0.70	n.s.
Error (b)	11	882.1	80.2		
2-min standing diastolic blood pressure					
Method	1	232.8	232.8	1.32	n.s.
Error (a)	11	1,937.4	176.1	2.64	n.s.
State	1	88.7	88.7	1.33	n.s.
M × S	1	29.7	29.7	0.45	n.s.
Error (b)	11	733.6	66.7		
Δ standing diastolic blood pressure					
Method	1	45.6	45.6	0.53	n.s.
Error (a)	11	952.2	86.6	1.09	n.s.
State	1	258.6	258.6	3.27	n.s.
M × S	1	21.7	21.7	0.27	n.s.
Error (b)	11	869.7	79.1		
Serum glucose, mg/100 ml					
Method	1	1,974.5	1,974.5	6.08	< 0.05
Error (a)	11	3,569.3	324.5	0.87	n.s.
State	1	5,700.9	5,700.9	15.37	< 0.005
M × S	1	1,563.1	1,563.1	4.21	n.s.
Error (b)	11	4,080.5	371.0		
Evans blue space, ml					
Method	1	25,206.2	25,206.2	0.11	n.s.
Error (a)	11	2,614,211.0	237,655.5	5.29	< 0.01
State	1	1,093,060.1	1,093,060.1	24.33	< 0.001
M × S	1	12,254.6	12,254.6	0.27	n.s.
Error (b)	11	494,262.8	44,933.0		

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES - Continued

(b) Analysis of variance data - Continued

Source	Degree of freedom	Sum of squares	Mean square	F	p
Red cell volume, ml					
Method	1	86,341.0	86,341.0	0.37	n.s.
Error (a)	11	2,587,081.6	235,189.2	22.32	< 0.001
State	1	88,045.0	88,045.0	8.36	< 0.025
M × S	1	105,324.1	105,324.1	9.99	< 0.01
Error (b)	11	115,916.4	10,537.9		
Blood volume, ml					
Method	1	204,849.1	204,849.1	0.28	n.s.
Error (a)	11	7,966,390.6	724,217.3	9.46	< 0.001
State	1	1,801,551.5	1,801,551.5	23.54	< 0.001
M × S	1	189,432.9	189,432.9	2.48	n.s.
Error (b)	11	841,842.6	76,531.1		
Hematocrit, vol %					
Method	1	2.052	2.052	0.12	n.s.
Error (a)	11	190.872	17.352	8.51	< 0.001
State	1	21.786	21.786	10.68	< 0.01
M × S	1	5.013	5.013	2.46	n.s.
Error (b)	11	22.431	2.039		
Serum Na, mEq/l					
Method	1	9.330	9.330	2.52	n.s.
Error (a)	11	40.759	3.705	0.79	n.s.
State	1	21.787	21.787	4.65	n.s.
M × S	1	0.824	0.824	0.18	n.s.
Error (b)	11	51.559	4.687		
Serum K, mEq/l					
Method	1	1.118	1.118	8.28	< 0.025
Error (a)	11	1.480	0.135	1.15	n.s.
State	1	0.615	0.615	5.26	< 0.05
M × S	1	0.336	0.336	2.87	n.s.
Error (b)	11	1.289	0.117		

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES - Continued

(b) Analysis of variance data - Continued

Source	Degree of freedom	Sum of squares	Mean square	F	p
Serum Cl, mEq/l					
Method	1	16.906	16.906	1.95	n.s.
Error (a)	11	95.503	8.682	2.15	n.s.
State	1	0.031	0.031	0.01	n.s.
M × S	1	11.692	11.692	2.89	n.s.
Error (b)	11	44.431	4.039		
Serum osmolarity, mOsm/l					
Method	1	23.762	23.762	0.39	n.s.
Error (a)	11	665.294	60.481	1.99	n.s.
State	1	346.385	346.385	11.37	< 0.01
M × S	1	1.878	1.878	0.06	n.s.
Error (b)	11	335.052	30.459		
Urine volume, ml					
Method	1	5,242.6	5,242.6	0.49	n.s.
Error (a)	11	116,837.9	10,621.6	4.64	< 0.01
State	1	499.9	499.9	0.22	n.s.
M × S	1	13,965.0	13,965.0	6.09	< 0.05
Error (b)	11	25,207.1	2,291.6		
Urine Na, mEq/l					
Method	1	3,213.1	3,213.1	0.78	n.s.
Error (a)	11	45,069.4	4,097.2	3.73	< 0.025
State	1	49,809.4	49,809.4	45.40	< 0.001
M × S	1	14,987.2	14,987.2	13.66	< 0.005
Error (b)	11	12,069.4	1,097.2		
Urine K, mEq/l					
Method	1	940.5	940.5	0.88	n.s.
Error (a)	11	11,755.0	1,068.6	1.05	n.s.
State	1	13,754.0	13,754.0	13.58	< 0.005
M × S	1	7.9	7.9	0.01	n.s.
Error (b)	11	11,142.1	1,012.9		

TABLE V.- CARDIOVASCULAR AND METABOLIC VARIABLES - Concluded

(b) Analysis of variance data - Concluded

Source	Degree of freedom	Sum of squares	Mean square	F	p
Urine Cl, mEq/l					
Method	1	3,799.374	3,799.374	0.94	n.s.
Error (a)	11	44,320.852	4,029.168	3.10	< 0.05
State	1	33,408.616	33,408.616	25.67	< 0.001
M × S	1	14,070.851	14,070.851	10.81	< 0.01
Error (b)	11	14,310.473	1,301.339		
Urine osmolarity, mOsm/l					
Method	1	220,519.940	220,519.940	5.90	< 0.05
Error (a)	11	411,101.600	37,372.870	1.49	n.s.
State	1	228,609.385	228,609.385	9.13	< 0.025
M × S	1	71,712.021	71,712.021	2.86	n.s.
Error (b)	11	275,480.600	25,043.690		

TABLE VI.- BODY WEIGHT CHANGES DURING THE EXPERIMENTAL PERIODS

	N1	H2							N3
Subject	kg	Pre wt, kg	End wt, kg	Δ wt, N1 - H2 end	Body wt loss, percent	Δ time, hr	Rate of wt loss, g/hr	Rate of wt loss, g/hr - m^2	kg
Acute group									
MM	76.395	75.280	73.655	2.740	3.59	4.93	329	167	75.445
HV	68.700	69.130	67.115	1.585	2.31	3.25	620	330	68.765
WL	73.610	72.225	70.640	2.970	4.03	3.12	508	267	72.030
RP	69.955	68.685	67.325	2.630	3.76	3.00	453	257	69.450
JM	65.055	65.025	63.020	2.035	3.13	3.35	598	340	64.720
$\bar{X}$	70.743	70.069	68.351	2.392	3.36	3.53	502	272	70.082
±SE	1.97	1.73	1.79	0.254	0.301	0.355	52.68	31.04	1.78
n	5	5	5	5	5	5	5	5	5
Chronic group									
DD	72.425	71.935	68.770	3.655	5.05	47.17	67	35	71.495
JB	66.340	68.215	65.115	1.225	1.85	48.63	64	37	67.470
JL	60.050	60.785	58.085	1.965	3.27	47.88	56	34	60.000
MP	66.485	68.450	65.280	1.205	1.81	47.20	67	38	66.860
DL	70.490	71.800	67.830	2.660	3.77	47.75	83	45	69.890
CO	73.975	75.025	71.360	2.615	3.53	48.53	76	40	74.180
FK	77.065	77.550	71.860	5.205	6.75	48.27	118	61	75.070
JG	78.085	78.125	74.735	3.350	4.29	46.18	73	37	77.730
$\bar{X}$	70.614	71.486	67.879	2.735	3.79	47.70	76	41	70.338
±SE	2.15	2.02	1.82	0.474	0.577	0.291	6.72	3.11	1.98
n	8	8	8	8	8	8	8	8	8

TABLE VII.- COMPOSITION OF METRECAL LIQUID*

Quantity, ml	915.2
Kcal	900
Protein, g	70
Fat, g	20
Carbohydrate, g	110
Vitamin A, U.S.P. units (palmitate)	5000
Vitamin D, U.S.P. units	400
Vitamin C, mg (sodium ascorbate)	100
Thiamine, mg (hydrochloride)	2
Riboflavin, mg	3
Niacinamide, mg	15
Calcium, g	2
Phosphorous, g	1.7
Iron, mg (ferrous sulfate)	10
Iodine, mg (sodium)	0.15
Vitamin E, Int. units (D-alpha-tocopheryl acetate)	10
Pyridoxine, mg (hydrochloride)	2
Vitamin B_{12}, mcg (cyanocobalamin)	2
Calcium pantothenate, mg	10
Sodium, g (iron pyrophosphate)	1.0
Potassium, g	2.3
Copper, mg (sulfate)	1.5
Manganese, mg (sulfate)	2

Other ingredients: Concentrated sweet skim milk, milk protein concentrate, sugar, partially hydrogenated soy oil, artificial flavor, 0.04 percent calcium cyclamate, natural color, chondrus extract, and calciferol

*Label values

TABLE VIII.- COMPOSITION OF METRECAL WAFERS

Quantity, no. wafers	36
Kcal	900
Protein, g	70
Carbohydrate, g	110
Fat, g	20
Vitamin A, U.S.P. units (palmitate)	5000
Vitamin D, U.S.P. units	400
Vitamin C, mg (ascorbic acid)	100
Thiamine, mg (mononitrate)	2
Riboflavin, mg	3
Niacinamide, mg	15
Calcium, g (phosphate)	1.1
Phosphorous, g	1.2
Iron, mg (sulfate)(carbonate)	15
Iodine, mg	0.15
Vitamin E, Int. units (D-alpha-tocopheryl succinate)	10
Pyridoxine, mg (hydrochloride)	2
Vitamin B_{12}, mcg (cyanocobalamin)	2
Calcium pantothenate, mg	10
Sodium, g	0.9
Potassium, g (carbonate)	2
Copper, mg	1.5
Magnesium, g (carbonate)	0.3
Manganese, mg (sulfate)	2

Other ingredients: Soy protein concentrate, wheat flour, sugar, calcium caseinate, molasses, corn oil, coconut oil, dried torula yeast, ammonium bicarbonate, cottonseed flour, lecithin, wheat bran, cornstarch, calciferol, cinnamon, lemon oil, and imitation butter and vanilla flavors

NASA-Langley, 1966 A-2206